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President's Column

Ian H. Gotlib, Ph.D.
Stanford University

Last month in The Journal of the American Medical Association, Ron Kessler and his colleagues published some of the findings from the recent National Comorbidity Survey Replication (NCS-R) study concerning the epidemiology of Major Depressive Disorder. The NCS-R is a nationally representative study of 9,090 people aged 18 and older. The results of this article were picked up by all of the major media. Television, radio, and newspaper reports focused on two

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Habit Learning and Psychiatric Disorders

Barbara Knowlton, Ph.D.
University of California at Los Angeles

Editor's note. Dr. Knowlton was one of the invited speakers at 2002 meeting of SRP.

One of the most compelling recent ideas in behavioral neuroscience is that there are different memory systems that depend on different brain systems. How do these different memory systems differ in terms of their behavioral properties? Clearly, we need a good understanding of the characteristics of the different memory systems in order to understand the operations performed by the brain substrates of these different systems. Learned behaviors underlie many types of psychopathology

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Members' Corner

Origins of Psychopathology Research: The Discovery of P300 and Cognitive Psychophysiology

Stuart R. Steinhauer, Ph.D.

The history and evolution of research in psychopathology and brain science is often lost in the morass of empirical data. The editors of the Newsletter suggested that it might be worthwhile to consider the background of how ideas and approaches that now seem obvious came to be established. For an initial glimpse, it seemed

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major findings from this new study. First, the one-year prevalence of diagnosable depression in the community is between 6 and 7 percent, which represents a slight increase over the last decade. Second, the number of Americans who are being treated for depression rose dramatically between 1987 and 1997, from 1.7 million to 6.3 million. That means that about 57 percent of depressed people are receiving treatment for depression. So the good news is that a much larger proportion of people with depression are now seeking and receiving treatment. We should be encouraged by this figure. As many of you know, we do reasonably well in treating depression: by receiving cognitive therapy, interpersonal therapy, or antidepressant medication, alone or in combination, approximately two-thirds of depressed individuals recover from the disorder.

But there is another, troubling, finding from the NCS-R. Kessler and his colleagues found that treatment met minimum standards of treatment adequacy, established by the Agency for Health Care Policy and Research, in only 21 percent of patients with recent depression. Almost 80 percent of patients who are treated for depression do not receive adequate care. As Kessler himself has said in interviews, "That's the most disturbing thing of all. After all these years of trying to get them in (to treatment)... we've screwed up." Kessler went on to note that many doctors may be unaware of treatment advancements, patients may be terminating treatment to early, and many patients may be offered unproven therapies.

It is difficult not to be discouraged by these findings. Discouraged, but not surprised. A decade ago, John Weisz presented data indicating that therapies practiced in community clinics do not come close to attaining the success rates reported in controlled clinical trials of the same treatments, demonstrating all too clearly that efficacy does not necessarily translate into effectiveness. I am aware that most members of SRP are not psychotherapy researchers; indeed, most SRP members are also not involved in the study of depression. We are, by and large, basic scientists who are concerned with understanding the processes and mechanisms underlying different forms of psychopathology.

Nevertheless, I think that all of us should be disturbed by the NCS-R findings. SRP members include the strongest psychopathologists in the country. Our annual meeting is a showplace for the best research in experimental psychopathology, investigations that elucidate factors involved in the etiology and maintenance of various forms of psychopathology. There is no question that findings from our studies have important implications for the treatment of psychological disorders. But research on basic processes in psychopathology is not tied tightly enough with the conceptualization and design of treatment studies, which should be incorporating findings from such investigations. And treatment studies, in turn, it appears, do not inform community practice as strongly as they should.

It sounds trite to say that we should do more to insure that basic research informs treatment. This has been said many times before. But I think that the NCS-R findings highlight the magnitude of this problem. I think that experimental psychopathologists, as a group, have been remiss in not making a greater effort to communicate findings from our studies to treatment researchers, and in not often enough elucidating specific implications of our studies for the treatment of disorders. And we have left it to treatment researchers to deal with the generalizability of their methods and findings to community samples. Rectifying this situation will force us to reach out beyond our usual environments and begin communicating findings in a way that may be more immediately useful. And perhaps more important, our research will be strengthened by examining whether, and under what conditions, the phenomena we believe are involved in various forms of psychopathology play a role in treatment and recovery from disorder. If all of us, including treatment researchers, make this outreach effort, our basic science will likely be strengthened, and we may begin to close the large gap between efficacy and effectiveness.

Barbara Knowlton, Ph.D.

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and in many other cases learning deficits associated with psychopathology contribute to functional incapacity. Thus, the study of the neural basis of different forms of learning is fundamental to the study of the biological basis of psychopathological syndromes.

In my laboratory, we are focusing on the contrast between the type of memory that depends on medial temporal lobe structures including the hippocampus, and the type of memory that depends on the striatum. Damage to structures in the medial temporal lobe leads to the amnesic syndrome. These patients have severe problems in learning new facts and events, although many studies have shown that they are able to normally learn a variety of other types of information, including motor skills and perceptual priming. The study of amnesic patients has provided us with a clear distinction between memories for facts and events, for which people are consciously aware that they are remembering this information, and the type of memories where learning is occurring without awareness of what is being learned. This distinction between "explicit" and "implicit" memory is shown by the former being dependent on medial temporal lobe structures and the latter being independent of these structures.

Unlike explicit memory, implicit memory does not depend on a single integrated brain system. Rather, different types of implicit memory depend on different brain

systems. These different forms of implicit memory may differ as much from each other as they do from explicit memory in terms of their properties. I have focussed much of my research on the implicit learning of habits. Habit learning refers to the implicit incremental learning of stimulus-response associations. The idea is that when reinforcement occurs, it serves to "stamp in" the association between the response and the eliciting stimulus. Reinforcement is required to forge the stimulus-response association, but it is not part of the learned representation. That is, when an organism is presented with the eliciting stimulus, it will automatically produce the response. The response is not produced in an effort to attain the reinforcer. These stimulus-response associations are learned implicitly, and thus one may not be necessarily aware of how or why a particular stimulus elicits a response. In everyday terms, we often think about our non-goal directed actions as being done out of "force of habit". We may find ourselves following our daily route to work before realizing that we had actually been planning to go elsewhere. I would assert that in this case, a particular intersection, for example, may automatically elicit a left turn response even if we intended to make a right turn. Several maladaptive behaviors such as compulsive drug taking or overeating could also be seen as "habits". These behaviors could persist even when the particular outcome or reinforcement is no longer desired.

A considerable amount of work using experimental animals has linked habit learning with the striatum. Many of the learning tasks that depend on the striatum appear to be learned gradually and incrementally as habits. For example, rats are able to learn to run down the lit arms of a maze to obtain food reward. The rats learn this response gradually across days, and they do not need a intact hippocampus to learn this response, suggesting that they are learning "implicitly" (if it is possible to use this term with a rat). However, a major problem in studying habit learning is that is not always apparent whether a particular behavior is a habit or not. It has sometimes been the case that habit learning has been defined circularly- if it depends on the striatum, it is a habit, and the mnemonic function of the striatum is habit learning. Of course, to move beyond this we need to be able to distinguish habits from other types of memory based on behavioral evidence. A key part of the definition of a habit is the idea that they are based on stimulus-response associations. Thus, they should not be affected by a decrease in the attractiveness of the reinforcer, since the reinforcer is not part of the learned representation. One way to probe this has been to train rats on an ostensible habit learning task, and then devalue the reinforcer by pairing it with the experience of illness. For example, if rats had been trained to run down lit arms of a maze for a sucrose pellet reward, we can then make the sucrose pellets unattractive by pairing them with exposure to a compound that induces illness. When presented with the sucrose, the rats now refuse it. The question is, will they still perform the "habit" of running down the arms as quickly and accurately as

rats who still like sucrose. We have shown that in fact they do, consistent with the idea that rats have learned a stimulus-response habit in this striatal dependent task. The light automatically elicits a running response. The sucrose merely served to strengthen this link during training.

If the role of the striatum is the formation of stimulus response habits, then it should be the case that other tasks in which stimulus-response associations are learned also depend on the striatum. For example, instrumental conditioning using an interval schedule has been shown to be insensitive to devaluation of the reinforcer. In this task, the subject needs to respond sometime during an interval to receive reinforcement. Reinforcement rate is not directly tied to the rate of responding. An example would be checking your mailbox for mail. If your mail is delivered on a daily interval schedule, you will not receive any more mail if you check your box 100 times per day than if you check once per day. We trained rats with striatal damage and control rats in a bar pressing task using an interval schedule of reinforcement. We actually found that both groups seemed to learn the bar pressing response fairly similarly. However, differences emerged when we devalued the reinforcer. As expected, control rats continued to press the bar at the same rate as their counterparts that did not experience devaluation. This result replicates a number of past studies. However, the rats with striatal damage actually stopped responding when they no longer found the reinforcer attractive. In a sense, they were behaving more insightfully than intact rats, in that they no longer performed a task that led to an unwanted outcome. Because their habit learning system was damaged, they may have learned the association between the response and the outcome explicitly.

The identification of a habit learning system that is dependent on the striatum has major implications for a number of human syndromes that have been linked to striatal dysfunction. For example when this system is overly active, one may see the type of excessive stimulus-bound behavior that is present in obsessive-compulsive disorders and addiction behaviors. Because these behaviors are not directly controlled by the desirability of the outcome, they may be quite persistent. Conversely, I would also argue that deficits in habit learning can have negative consequences. Although the information we learn as habit can be readily learned as explicit facts, there is a cost involved in over-reliance on explicit memory retrieval. Habits are performed automatically with little demand on cognitive resources. In contrast, explicit memory retrieval requires a fair amount of cognitive resources. Because cognitive resources are limited, it is useful that over-learned, routinized behaviors can be performed without drawing on these limited resources. If striatal dysfunction leads to a deficit in habit learning, patients may exhibit overall declines in executive function due to a need to allocate cognitive resources to perform tasks that would normally not require these resources. For example, there is evidence that patients with schizophrenia have deficits in automating skills, which may be linked to striatal dysfunction.

Although this deficit may not be a core feature of the disease, it seems quite possible that is would contribute to lowered functional capacity in these patients. A key area in translating the laboratory research on habit learning into clinical studies is to identify real-world examples of habit learning that we can study in patients with striatal abnormalities.

Reading List:

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Knowlton, B.J. (2002). The role of the basal ganglia in learning and memory. In Neuropsychology of Memory, Third Edition (L.R. Squire & D. L. Schacter, eds.) Guilford Publications, New York, pp143-153.

Packard, MD & Knowlton, BJ (2002) Learning and memory functions of the basal ganglia. Annual Review of Neuroscience, 25, 563-593.

Tekin, S. & Cummings, J.L. (2002). Frontal-subcortical neuronal circuits and clinical neuropsychiatry: an update. Journal of Psychosomatic Research, 53, 647-654.

KUDOS!!!

Congratulations to:

• Elaine Walker, on being named a Fellow of the American Psychological Society.

MEMBERS' CORNER

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intriguing to review the "discovery" of the P300 component of the event-related brain potential. P300 is broadly defined as a scalp-positive electrical wave recorded from the, about one-third of a second after an event, which has been shown to be related to a wide variety of complex processing events, involving meaning or salience, transformation of information, identification of event characteristics, and many more. The reduction of P300 amplitude in schizophrenia is perhaps the most widely replicated experimental phenomenon in schizophrenia, and P300 is deviant in a number of other disorders. Its discovery in 1964 (published by Sutton et al. in 1965) arguably initiated the main thrust of cognitive psychophysiology. The story behind this event, however, is rooted firmly in behavioral psychopathology research.

Schizophrenia researchers had used measures of reaction time to study attention and processing differences dating back to the time of Kraepelin (for reviews of the RT literature in schizophrenia, see the excellent paper by Nuechterlein in Schizophenia Bulletin (1977), including in depth commentaries in the same issue, and an update by Rist and Cohen (1991)). A systematic examination of reaction time slowing to auditory and visual stimuli, with dependencies on timing and sequences of stimuli, was conducted by Shakow and colleagues at Worcester State Hospital as early as 1937 (see Shakow, 1972). Much of this work focused on preparatory intervals,

and effects of regular as compared to irregular series of stimuli.

In a varying approach to RT in schizophrenia, Sutton and colleagues had been studying the effects of sequences in which the sensory modality of the stimulus was changed. In the initial study, subjects responded to either a red or green light, or high or low pitched tone, presented in an irregular series, with a simple finger lift. In general, RT was found to be slower when the modality of the stimuli shifted between stimuli, as compared to repetition of stimuli in the same modality. What was particularly interesting was that the effect of this shift in modality resulted in significantly slowed RTs for the schizophrenic patients, especially with shifts from light to sound stimuli (Sutton et al., 1961). This "cross-modality" effect continued to be observed in a number of subsequent studies.

At this point in the story, technology and serendipity arrive. Samuel Sutton was heading the Psychophysiology Section of the Biometrics Research Program, which Joseph Zubin had created at New York State Psychiatric Institute in upper Manhattan. But space for the Psychophysiology and the Verbal Behavior sections was scarce, when the Director of Brooklyn State Hospital (now renamed Kingsborough Psychiatric Center) offered access to patients, and a series of rooms in the basement of the main building. In those dark, dungeonlike hallways, with rust stains along the concrete floors, a series of experimental laboratories were established: Mitchell Keitzman, studying visual temporal integration; Gad Hakerem, investigating pupillary dilation and cognition; and Samuel Sutton (joined by Patricia Tueting), using the EEG and recording sensory evoked potentials.

Sutton was experienced in electrophysiology (originally, with cats). He was able to borrow EEG amplifiers from E. Roy John, who had been a student with him at the University of Chicago. In Hakerem's pupillography lab across the hall, there was a CAT – not the purring kind, but a Computer of Average Transients. In the days before digital computers, the CAT enabled physiological signals to be "averaged" by summating briefly sampled voltages; averages were computed by using a voltage. The CAT spent much of its time in transit across the hallway between the two labs.

Although the laboratories were in Brooklyn, Sutton lived in Manhattan (and preferred not to drive), Zubin in New Jersey, and Hakerem slightly upstate in Rockland County. This resulted in a good deal of car pooling to reach the Brooklyn labs, and lots of conversation. According to Zubin (and verified by Gad Hakerem, who is still an active psychophysiologist and was in the car), one of these discussions centered on the ability to begin to record evoked potentials. Zubin asked Sutton whether it might be possible to know what was happening in the brain in response to the cross-modality presentation. Sutton initially responded that they could even do it that afternoon. It would be easy to look at the effects of changing the sequences of a simple light or sound.

Then complexity struck – Sutton realized that little was known about how motor responses might "contaminate" the scalp recording, and he was really interested in possible slight modifications of the sensory response due to modality changes. Of course, healthy subjects needed to be studied first. He decided to omit a button press, but was still concerned that the subject needed to do something to demonstrate attention. He introduced the simple notion that the subject would overtly guess, before each critical stimulus, what the next stimulus would be. A trial involved a cue stimulus followed 3-5 seconds later by the test stimulus. Sometimes the cue indicated that the test stimulus would definitely be a light or a click (a certain condition), but other times the cue indicated that the test stimulus was unknown (uncertain). Thus, critical comparisons could be made between responses to uncertain vs. certain stimuli. For the initial experiments, the pairs used were sound-sound, light-sound, and light-light. That is, a sound cue always predicted a sound, but a light cue could be followed by a light (which happened most of the time) or by a sound. Remember that the RT phenomenon of greatest interest involved shifting attention to a sound after a light had been presented. However, the sound after a light is also an unpredictable event.

A memo dated May 25, 1964, reported initial findings briefly to Zubin: "Note that the evoked potential to sound after light is different in waveform and larger in amplitude. If these waveforms, as we now suppose, are not primary sensory responses, but are in fact either cortico-cortuo [sic]; or cortico – subcortical (e.g. reticular formation) they could represent an orienting or alerting component of sensory response. Extending this line of thought, the larger evoked potentials relate to the surprise of the subject and in reaction time give longer reaction time".

The waveform mentioned was larger than all of the earlier sensory components that had been established in the literature. It was positive at scalp, using a linked ear reference, and the peak amplitude occurred at approximately 300 ms. The larger amplitude of the response to the uncertain stimuli, since termed P300, became the focus of the research. Rather than concentrate on the original aspect of interest (modality change), degree of uncertainty, event probability, as well as outcome of the prediction – right vs. wrong – were large differences that were observed and became major variables in subsequent studies. Many years later, Sutton wrote to Zubin while preparing a review of the original findings "We did not run the modality shift control till May 29th and as you know we discovered a relatively small effect due to modality shift. The big effect was due to uncertainty" (Sutton, May 9, 1978). In the original data, the increased amplitude to resolution of uncertainty was replicated in 36 of 36 comparisons in the initial eight subjects.

One would think that such a finding would be accepted readily – but the initial paper met with hostility; one reviewer found the likelihood that such psychological complexity could be detected in what was the largest electrical deflection in the average

waveform to be unlikely, and suggested that it was just an artifact. (Actually, Sutton always treated any new finding the same way – first try to determine if there was a technical mistake before claiming that you have something real). The paper was eventually published in Science (1965), and a second paper two years later, with Patricia Tueting (Sutton et al., 1967), demonstrated that the P300 component could be elicited even when a significant event failed to occur – in essence, it was an endogenous potential that did not depend on sensory stimulation.

These two papers resulted in a focus on cognitive psychophysiology – as Sutton would later emphasize, combining stimulus characteristics, physiological evaluation, and behavioral involvement of the subject. Much of the work now turned to exploring P300 and related ERP components in normal subjects. In 1972, schizophrenia reentered the picture, with studies by (Walton) Tom Roth and Cannon, and by Robert Levit in his dissertation in Sutton's lab (published as Levit et al., 1973), indicating clear reduction in P300 amplitude for schizophrenia patients for the first time. Since then, literally thousands of investigations of P300, slow wave, scalp negativities, and other associated phenomena have been recorded in healthy subjects and patients.

The rigor and replicability of those first studies was noted in a retrospective of those P300 papers (Bashore and van der Molen, 1991). Not only was cognitive psychophysiology established as a valuable method for studying normal and abnormal processes, but special emphasis on the consequences of cognition and behavioral involvement of subjects became pervasive in psychopathology research. As Sutton once mentioned to me "the grammar of the brain" had become directly amenable to understanding. And that same CAT now purrs away in retirement, sitting in a corner of my laboratory in Pittsburgh.

(copies of the original Science papers are available, by permission of AAAS, at http://www.wpic.pitt.edu/research/biometrics/labpubs.html).

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Nuechterlein, K.H. (1977) Reaction time and attention in schizophrenia: A critical evaluation of the data and theories. Schizophrenia Bulletin, 3: 373-428.

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Sutton, S., Hakerem, G., Zuibn, J, and Portnoy, M. (1961). The effect of shift of sensory modality on serial reaction time: A compa4ison of schizophrenics and normals. American Journal of Psychology, 74: 224-232.

Sutton, S., Tueting, P., Zubin, J., & John, E.R. (1967). Information delivery and the sensory evoked potential. Science, 155, 1436-1439.

TORONTO HOSTS SRP

Dick Steffy, Ph.D., University of Waterloo

The city of Toronto, Ontario, Canada welcomes delegates to the SRP 2003 Annual Meeting convening in the Toronto Marriott Eaton Centre Hotel from October 16th through 19th, 2003. Under the leadership of President Ian Gotlib, an exciting program has been planned by Sheri Johnson and will fit our traditional format. Special addresses from President Gotlib, from William Iacono, Sir Michael Rutter (Zubin award winner) and Steve Suomi will accompany poster sessions, symposia, and our typical lavish banquet. Take note please that the Marriott Hotel's location dead center in the life of downtown Toronto, so extracurricular opportunities are abundant.

Toronto is a cosmopolitan city of 2.5 million people with a large tourist industry. It sports four English speaking newspapers and a substantial multicultural quality of life. Forty-three percent of the population represent diverse racial minorities and one can hear more than a hundred different languages or dialects in the city. Millions of tourists each year recognize the city's rich diversity.

Of convenience to visitors and residents alike, Toronto has the second largest public transit system on the continent and an exceptionally large underground pedestrian system (a thousand stores and restaurants when one goes down into the catacombs). Of particular importance to any visitors, Toronto is rated as one of the safest large metropolitan area on the continent.

Toronto is a fun place. Although one senses a large city all around, it is not a place of claustrophobia. The buildings are large but not densely packed. Of importance to city-goers and visitors alike, Toronto lacks zones of transition. Within a block or two of the major business and transit corridors are networks of narrow residential streets with old country charm. One does not feel hurried and hassled in this city. Onlookers see many attractive features. For example, the Marriott Hotel is next to the city hall, and gives easy sight of the CN Tower (one of the largest freestanding buildings in the world). Within a half-mile radius there are several major ball parks that host professional teams (the Blue Jays, the Raptors, the Maple Leafs [playing international baseball, basketball and hockey respectively] plus a Canadian

league football team named the Toronto Argonauts). Art galleries, boutiques, live-action theatres (in October featuring Grease, the Philadelphia Story, The Lion King, Mamma Mia and various off-Broadway attractions), a concert center, and a wide range of restaurants are sprinkled throughout a walkable area. This is a picturesque lakeside city (a 20 minute walk to the shore of Lake Ontario) with a warm and accommodating quality, and it is widely recognized as a great location for conventions both large and small (APA is meeting in Toronto this summer) because of its ambience and phenomenal currency exchange rates favoring its U.S. visitors.

The Marriott Hotel is connected directly to the Eaton Centre shopping complex which is perched on the major street of the city (Yonge Street). The hotel has a quite good quality of meeting rooms that will cover all of our requirements, with 459 guest rooms and 24 suites. There is a small rooftop pool, whirlpool, sauna, fitness center, two restaurants, two lounges and other amenities. For your information, the hotel entrance is located on Bay Street, approximately 15 miles from the airport. There is a limousine service at the airport to carry passengers into the city core along fast access roadways.

Although I cannot guarantee the climate, one can ordinarily expect a relatively balmy fall day in mid-October with requirements for at least a sport coat in the evening hours. Is snow possible? Yes it is, but improbable for most mid-October times. Do not, however, expect sweltering heat.

All are concerned these days about the SARS epidemic. As you probably know, the World Health Organization caution against travel to Toronto has been lifted. By the time we meet, we anticipate that APA and other large groups will have had successful conventions in the city. There are currently a few active cases in the city, but what is most comforting is the fact that all active cases are located within the health care system itself. Rigorous quarantines have made the city free to operate in normal ways. You may encounter some questions about your respiratory health as you reach the airport, but you are unlikely to recognize any other encumbrances.

Exact information about registration routines and conference registration will be circulating to you in the next month or so. If you have questions at any time about the arrangements, feel free to write or call Dick Steffy at the University of Waterloo. My email is steffy@uwaterloo.ca, my phone is 519-888-4567 Ext. 2548.

We hope to host you at these exciting meetings in mid-October.

A Note from the Editor.

Toronto sounds great, doesn't it!?! Fellow SRP members, you might want to add on an extra day or two to your travel plans, so that you can take advantage of some of what Toronto has to offer, as well as attend what is in all likelihood going to be an excellent meeting.

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